

sequence beginning with 50 Joules (J), until success (i.e. atrial defibrillation threshold). Delivered energy, peak current, and impedance were obtained at atrial defibrillation threshold for each patient. **Results:** Mean energy and current requirements for atrial defibrillation were reduced with E while impedance was similar to placebo (see table). In addition, 1 patient receiving E spontaneously converted to sinus rhythm prior to electric shock and another patient, also receiving E, failed cardioversion at 360 J \times 2 attempts. **Conclusions:** Among patients who were successfully cardioverted, esmolol significantly reduced energy and current requirements for atrial defibrillation. Esmolol may be a useful adjunctive agent for atrial cardioversion.

1001 Ventricular Arrhythmia and Syncope: Clinical Aspects

Tuesday, March 18, 1997, Noon-2:00 p.m.
Anaheim Convention Center, Hall E
Presentation Hour: Noon-1:00 p.m.

1001-77 Differentiation of Right Ventricular Outflow Tract and Left Basal Ventricular Tachycardia: Electrocardiographic Criteria

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Ventricular tachycardias (VTs) with a LBBB pattern in lead V1 and tall, monophasic R-waves in the inferior leads usually are ablated successfully from the right ventricular outflow tract (RVOT), but exceptions have been reported. We identified 29 patients who underwent radiofrequency catheter ablation (RFCA) between 1/94-6/96 with this VT morphology. Nineteen patients had VTs successfully ablated in the RVOT. Ten patients (8 female/2 male; ages 13-52 years), despite clinical characteristics similar to the other 19 patients, had VTs mapped to regions outside the RVOT. Nine of these 10 patients had structurally normal hearts; one had a dilated cardiomyopathy. When compared to the 19 VTs successfully ablated in the RVOT, the ECGs of these 10 VTs had earlier transition zones (median = V3 vs. V5; $p < 0.001$), slightly more rightward axes (90 ± 4 vs. 83 ± 5 ; $p = 0.002$) and a small R-wave in V1 ($10/10$ vs. $9/19$; $p = 0.011$). All 10 VTs were mapped (activation/pacing) in both the RV and LV. No attempt at RFCA in the RVOT ($N = 9$) eliminated VT while RFCA in the anterobasal LV ($N = 6$) eliminated 2 VTs.

Conclusion: An early transition zone, rightward axis and R-wave in V1 suggest that VT arises outside the RVOT, despite a LBBB pattern in V1 and tall, monophasic R-waves in the inferior leads. Mapping and limited RFCA successes suggest that some of these VTs arise from the LV outflow tract or other basal LV foci. Recognition of these electrocardiographic variations may help advise patients and direct one's approach to RFCA.

1001-78 Ventricular Tachycardia in a Closed-chest Ovine Model in the Chronic Phase after Myocardial Infarction

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In most animal models ventricular tachycardia (VT) can be initiated mainly in the acute and subacute phase of myocardial infarction (MI). Thereafter the incidence of VT induction often declines and many episodes become nonsustained or ventricular fibrillation (VF) is induced. The study purpose was to develop an animal model of sustained VT in the chronic phase of MI. To create MI, balloon-occlusion of the left anterior descending coronary artery for 150 minutes was performed in 14 sheep. Programmed stimulation (PES) was performed three to six weeks after MI using up to four extrastimuli at twice diastolic pacing threshold. During acute MI, four animals died. Sustained VT could be reproducibly induced in 8 of the 10 surviving sheep. Induced VTs were monomorphic and hemodynamically tolerated. The mean VT cycle length was 225 ms (170-300 ms). There were between one and three VT morphologies in individual animals. The overall number of induced VT episodes was 265 (5 to 70 episodes per animal). VF was never induced during PES. The duration of VT episodes varied between 30 seconds and 15 minutes. Most episodes were terminated using antitachycardia pacing or low-energy cardioversion. Postmortem evaluation showed transmural infarction with total replacement of myocardium with mature collagen tissue, but surviving subendocardial myocardium and Purkinje fibers were observed in most infarcted areas. This animal model provides an anatomic substrate similar to human MI with a high induction rate of sustained monomorphic VT in the phase of healed MI.

1001-79 Diverse Effects of Spontaneous Coronary Reperfusion on Ventricular Arrhythmia in Acute Myocardial Infarction

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We investigated the association between early intermittent closure and re-opening of infarct artery (IRA) and subsequent incidence of ventricular arrhythmia in patients with acute myocardial infarction (AMI). Fifty five patients with AMI receiving thrombolysis (I/V Duteplase 0.30-0.60 MU/Kg), within 6 hours of onset of symptoms were studied. Intermittency was defined as ≥ 2 episodes of resolution of elevated ST segment lasting ≥ 1 min on Holter ST segment recording started on admission. All patients received i.v. heparin following lytic therapy. The incidence of arrhythmia was calculated by dividing the 24 h Holter recordings in two phases: first 6 hours (early phase) and last 6 hours (late phase). Intermittency was documented in 49% (Group 1) and was absent in 51% (Group 2) of patients. The two groups were comparable regarding age, delay in treatment from the onset of symptoms, re-canalisation time and patency of IRA. The results were:

I. Incidence of arrhythmia during Early Phase

	Group 1	Group 2	p value
Isolated PVCs	476 \pm 164	98 \pm 38	0.014
Couplets	21 \pm 2	10 \pm 1	0.013
Ventricular Tachycardia	18 \pm 3	8 \pm 1	0.002

II. Incidence of arrhythmia during Late Phase

	Group 1	Group 2	p value
Isolated PVCs	11 \pm 5	165 \pm 84	0.034
Couplets	2 \pm 1	8 \pm 5	0.01
Ventricular Tachycardia	1 \pm 5	5 \pm 4	0.03

Values are mean \pm SD

Thus, spontaneous intermittent recanalisation represents an unstable state with increased incidence of ventricular arrhythmia during early AMI. Delayed ischaemic preconditioning may be a mechanism responsible for reduction in late phase arrhythmias.

1001-80 Differences in Circadian Variations of Ventricular Tachycardia and Premature Ventricular Complexes

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Circadian variations in the frequency of sustained ventricular tachyarrhythmias (> 30 s) (VT-f) and PVCs (PVC-f) are associated with changes in heart rate (HR). To determine if PVC-f could be used to predict the onset of VT, we examined common and differential characteristics of VT-f, PVC-f and HR. **Methods:** Holter ECGs from 53 patients with recorded spontaneous sustained ventricular tachycardia screened for ESVM trial were used to obtain circadian distributions of VT-f, PVC-f and HR. PVC-f and VT-f were normalized by individual 24 hr averages, and harmonic regression was applied to fit the experimental data using least squares ($r = 0.68-90$, $p < 0.001$). **Results:** High correlation was observed between PVC-f & HR and VT-f & HR ($r = 0.79$ and 0.75 , $p = 0.000004$ & 0.00003 , respectively), whereas correlation between PVC-f and VT-f was low ($r = 0.39$, $p = 0.06$). VT-f and HR had two peaks which were close to each other, however the peaks of VT-f (9 am & 6 pm) occurred earlier than those of HR (10 am & 7 pm) and corresponded more closely to periods of the maximum derivative of HR. PVC-f had multiple peaks from 8 am to 8 pm which were distributed during the day and did not correspond to that of VT-f. **Conclusions:** Circadian variations of sustained VT and PVCs are related to those of heart rate. Both VT-f and PVC-f are diminished at night, when HR is lowest. However, during the day, the highest frequency of VT corresponds to periods of maximum derivative (rate of increase) in HR, whereas the number of PVC has multiple low amplitude peaks.

1001-81 Lack of Endothelin-1 Rise May Contribute To Tilt-induced Syncope

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The nature of cardiovascular events leading to hypotension and bradycardia in patients with a positive tilt test remains unclear. In addition to sympathetic activation, endothelin-1 (ET-1) may also play a role in vasomotor tone. The changes in ET-1 release with the simultaneous measure of other vasocon-